

GASTRITIS (ABOMASTITIS)

⌘ **DEF:** inflammation & mucosal damage manifested clinically by → vomiting, Abd. Pain & hemorrhage from stomach.

⌘ Gastritis associated ē enteritis in a syndrome called gastroenteritis.

Gastro mucosal barrier:

1. PGE2:	2. Phospholipid	3. mucous & bicarbonate	4. Epithelium
☐ Enhance mucosal barrier. ☐ ↑ Mucous/ bicarbonate layer. ☐ Mucosal repair. ☐ VD → ↑ bl. flow → ↓ focal necrosis in pet A. ☐ Prevent inflammation.	initiated by PG 	☐ only in <u>glandular part</u> (so its <u>not</u> easily penetrated) ☐ only penetrated by <u>NSAIDs</u> for long dose as kataflam → ↓ PGE2	Glandular epith. Sq. <u>non glandular epith</u> (easily penetrated by any stress factor). <u>Horse stomach</u> → divided into glandular & non glandular part () them ↓ acidic area or farrow called <u>margophicatus</u>

PGE2:

- Constitutive → responsible for mucosal integrity - by COX1
- Inducible (inflammatory) → rise temperature - by COX2.
- Non selective NSAIDS → destructive.
- Selective NSAIDS → anti COX2 → not real but ↓ destruction.

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Mucosal protective	Mucosal aggressive (destruct)
1. Bicarbonate. 2. Phospholipids. 3. PGE2. 4. Blood flow. 5. Nitric acid.	1. HCL. 2. Pepsin. 3. Bile acid. 4. volatile FA - organic acids - valeric acid - 5. <u>Helicobacter pylori</u> → gastric ulcer & carcinoma.

- ❑ Normal daily HCL cross barrier & mucosa but blood flow rapidly remove.
- ❑ Equilibrium () mucosal protective & aggressive → healthy tissue.
- ❑ ↑ Mucosal aggressive & ↓ protective → ↑ time of exposure → desquamation of gastric epith. → Ulcer.
↓
- ❑ 1st reaction →
 - ↑ Motility (cramps - abdominal pain) → vomiting.
 - ↑ Secretion of mucous to protect mucosa → delay digestion (but m.o hide in mucosa & fail to putrefact)
- ❑ 2nd reaction (submucosa) →
 - Extravasation of bl. from cap. (bleeding)
 - Degranulation of mast cells → ↑ histamine release → local inflammation & edema & ++ parietal cell to secrete HCL. (the reaction)
 - ++ Dendritic cells (nerves) → pain.
 - Pain d.t → Hypermotility & ++ of dendritic cells.
- ❑ Chronic cases →
 - 1. ↑ Mucous → putrefaction.
 - 2. Chronic pain.
 - 3. Distension (no response to eating).
- ❑ Stress → ↑ cortisone → stop PGE2 → breakdown of gastric mucosa.

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Internal Medicine (Alimentary tract)

✓ Horse most labile by:

1. Abrupt exercise →

- Delay gastric emptying → ↑ contact () acid & gastric mucosa
- ↑ Intragastric pr. → push HCL from glandular part to non-glandular part → ulcer.

2. Phenylject (cheap anti-inflammatory): (Can be withheld 4-1)

- ☐ Normal dose → 2 mg/kg.
- ☐ Over dose → ulcer & gangrene.

Etiology:

a) Physical causes:

1. Ingestion of damaged food as rancid, spoiled food stuffs, coarse fibers
2. Ingestion of foreign material as bones, ropes esp. in pet animal
3. Trichobezoars (hair ball); esp. in long haired dog
4. Ingestion of some plants as in pet animals
5. Over eating → impaction, dilatation and 2nd gastritis → due to ↑ waste products by fermentation

b) Chemical causes:

1. Ingestion of irritants as cobber sulphate
2. Ingestion of fungal toxins
3. Acute lactic acidosis due to engorgement of CHO rich food → ruminitis and abomasitis
4. NSAIDs drugs as voltraine for long time
5. Allergic reaction → ↑ histamine → gastritis

c) Infectious causes:

1. Bacterial infection → helicobacter bacteria
2. Viral agents → BVD / cattle plague
3. Fungal agents → aspergillus spp.
4. Parasitic agents → gastrophylus equi larvae
5. In pet A → uremia, liver disease, neurological diseases, shock, sepsis, stress play a role in etiology of acute gastritis.

Clinical finding of gastritis:

Acute gastritis	Chronic gastritis
1. Vomiting from time to time in pet A. 2. Inappetance or complete loss of appetite. 3. Signs of abdominal pain. ▪ A layout & get out frequently ▪ Grunting sound in pet A & abnormal posture 4. Dehydration & Diarrhea + hypokalemic hypochloremic metabolic alkalosis (NaCl 0.9% - ringer- glucose) 5. Rancid smell during breath 6. Melena or black tarry stool 7. Vomit & blood 8. Position (praying position) of relief	1. No vomiting 2. Less severe than acute 3. Sub-acute Abd. pain 4. Partial loss of appetite 5. Sporadically vomiting 6. Dehydration sporadically

Diagnosis:

1. Case history
2. Physical examination
3. Lab. Analysis of gastric juice for pH, blood contents, bile contents

4. Differential diagnosis from:

- **Gastric dilatation** → vomiting is profuse & projectile without retching
- **Esophageal obstruction** → rapid vomiting after eating & vomitus is neutral (little), not have rancid smell
- **Intestinal obstruction** → alkaline vomitus with bile
- **Systemic toxin** → signs of toxicity

Treatment:

1. Removal of initiating agents.
2. Provide proper conditions to promote mucosal repair,
3. Correct 2nd complication as Abd. pain & vomiting
4. **Restriction of diet** is the initial therapy → as feeding may cause
 - Gastric distension
 - ↑ motility
 - ↑ vomiting
 - ↑ acid secretion
 - ↑ source of infection
5. **fluid & electrolyte** 40-60 ml/kg B.wt/day 3 times
6. **Antiemetic drugs** esp. in pet Å as **chlorpromazine** 0.1-0.5 mg/kg B.wt
7. In horse, **gastric lavage** to remove irritant chemicals

GASTRIC ULCER = ABOMASAL ULCER (PEPTIC ULCER):

Def:

- Acute or chronic **circumscribed mucosal defects** that extend through muscularis mucosa & have a **firm, raised margin**
- **Accompanied by** → anorexia, Abd. Discomfort, constipation or diarrhea, gastric hemorrhage & melena.
- Occur in **foals**, yearling, adult horse
- calves & adult cattle, but it is of **low incidence** than pet Å
- **↑ incidence in horse** with clinical problems as colic, poor appetite, poor body conditions

Etiology:

<u>1) In cattle:</u>	<u>2) In horse:</u>	<u>3) In pet A:</u>
☐ Long standing case of <u>vagus indigestion</u>, especially if abomasum is greatly involved ☐ Accidental ingestion of irritant of chemicals. ☐ Cattle plague ☐ Oral administration of heavy prolonged course of <u>antibiotics & arsenical preparation</u>	☐ Accidental ingestion of corrosive, chemicals ☐ Neglected case of gastritis ☐ Gastric squamous cell carcinoma ☐ Parasitic gastritis as <u>bot fly (gastrophilus)</u> → superficial ulcer. In horse, <u>habronema</u> ☐ Prolonged use of <u>NSAIDs (phenyl butazone)</u> → deep ulcer.d.t → Topical irritant effect of drug on epith. → Impairment of barrier properties of mucosa → Suppression of gastric <u>PGE2</u> synthesis → ↓ blood flow to gastric mucosa → Interference with the repair of superficial injury	☐ More common. ☐ <u>HCL, pepsin</u> is prerequisite for development of peptic ulcer ☐ <u>Stress ulceration</u> with erosion of mucosa and extend to muscularis ☐ <u>Systemic mastocytosis</u> - histamine is released with resultant gastric lesion ☐ <u>Female > male.</u>

Risk factors of gastric ulcer:

1. Exercise in racing horse → stress → ↑ gastric ulcer
2. Nervous horse are at greater risk
3. Stress
4. Administration of NSAIDs as phenyl-butazone
5. Colic with gastric lesions
6. Diet and feeding practices
7. Feed withholding cause gastric ulcer in horses, due to lack of buffering of acid produces during periods when the stomach is empty

Pathophysiology of gastric ulceration:

- ❑ Ability of stomach to withstand concentrated HCL without cellular damage called (**gastric mucosal barrier**)
- ❑ **The glandular epithelium** has mucus bicarbonate barrier, prostaglandins, cellular restitution & mucosal blood flow to protect itself from peptic injury
- ❑ **Mucosal blood flow** provide oxygen, nutrition to cells & remove excess hydrogen ions
- ❑ **Physiological level of corticosteroids** maintain adequate blood flow during streaming, plus blood flow supported by prostaglandin that ↑ mucus & bicarbonate production
- ❑ **Mucosal defense mechanism impaired by:**
 1. **NSAID** affect on formation of prostaglandin, So ↓ blood flow & gastric mucus and NaHCO_3 production
 2. **Corticosteroid** → ↑ gastric acid production & ↓ prostaglandin formation so persistence of gastric ulcer
 3. **Spinal insult** → contribute development of disease by alteration (gastric circulation & motility)
 4. **Shock** → ↓ blood flow to stomach → ↑ acidosis → ↓ delivery of bicarbonate ion to the surface cells
 5. **Uremia** → diffuse vascular injury to mucosal lamina propria with subsequent epithelial cell damage from anoxia
 6. **Chemical insult** to gastric mucosa comes from endogenous compounds (HCL), bile acids and pancreatic enzymes or exogenous HCL, toxin
- ❑ **Normally** → there is minimal leakage of secreted acid back into mucosa (very slow rate) & acid rapidly removed by vascular system
- ❑ **The exogenous, endogenous causes** → alteration in anatomical barrier so ↑ mucosal permeability to acids
- ❑ **↑ entry of acids** → direct damage to mucosa → destruction of sub epithelium and damage of bl. vessels → extravasation of blood and plasma
- ❑ **↑ acid in contact with mast cells** → degranulation of mast cells and release of histamine → ++ parietal cell secretion of HCL & local inflammation and edema

Gastric ulcer in horses:

- ☐ **Exposure of squamous mucosa to acid** → development of ulcer in most horses
- ☐ The horse is a continuous, variable HCL secretor, pH of equine gastric contents in the pylorus & antrum is less than 2 gastric pH is lowest, & acidity highest, when horses have been deprived of feed or have voluntarily stopped eating, often for as little as 2 hrs resulting in severe ulceration in gastric squamous epithelial mucosa
- ☐ **Exercise** alters eating behavior & **↑ gastric acid production** and splashes the gastric contents onto the less protected squamous portion of the stomach. It also **↓ blood flow to the stomach** lining and **delays gastric emptying**
- ☐ During Exercise intra-gastric pressure ↑ from approximately 12 mmHg at rest to as high as 50 mmHg, stomach volume decreases & the acidity of fluid within the proximal part of the stomach declines from 5-7 to 2-4
- ☐ The combination of reduced blood flow and exposure to low pH → mucosal damage, loss of protective mechanism & development of gastric mucosal lesions
- ☐ **Exercise** → **↑ intra-abdominal pressure** → **gastric compression pushing acid content in the proximal stomach**
- ☐ Other factors including physical injury to gastric mucosa, reflux of bile acids from duodenum and presence of volatile F.A. in stomach → all may contribute to development of gastric lesions
- ☐ All these factors → gastric ulcer → **reflux stimulation of pylorus** → **pylorospasm with ↑ gastric movement ↑ dilatation of stomach, abd. distension** → stop gastric emptying → obstruction → perforation → peritonitis (d.t leak of HCL)
- ☐ Ulcer extend to bl. vessels → rupture → hemorrhage → vomitus tinged with blood and melena
- ☐ Ulcer extend to cause stomach perforation → peritonitis and spleen involve

Clinical finding of gastric ulcers:

1. majority of horses with ulcer don't have clinical signs
2. Poor performance, refuse eating, fussy eating → not consume all food at a constant rate
3. Abd. pain
4. Melena, vomitus tinged $\bar{e}$ blood
5. Horse $\bar{e}$ sever gastric ulcer → with sever reflux esophagitis
6. Rupture of gastric ulcer, perforation, peritonitis
7. Intermittent diarrhea with pasty & scanty feces
8. Involvement of spleen with gastric ulcers in horse → rare
9. Pain on deep palpation over left flank
10. leukocytosis with a left shift

Diagnosis: (Tentative diagnosis) not observable & not characteristic. Depend on:

- 1) Presence of age related characteristic signs
- 2) Endoscopic (gastrosceop) → must be in horse. (2 meter in adult horse & 110-140 cm in foal)
- 3) Biopsy → H. pylori (may lead to cancer).
- 4) Response to treatment
 - It signs d.t 1ry ulceration → dramatic improvement within 1-5 days.
 - If no improvement → gastric ulceration → 2nd problem.

Treatment of gastric ulcer (anti ulcer therapy):

Goals:

1. Healing of ulcer
2. Prevention of ulcer recurrence
3. ↓ pain
4. ↓ gastric acidity
5. Protections of mucosa

1) Acid suppression:

1. Cimetidine & ranitidine (H₂ receptor blocker)

- ☐ Cimetidine (Tagamet) → less potent & cause nervous manifestation (not preferred) → 6.6 mg/kg B.Wt 3 times daily orally or IV injection.
- ☐ Ranitidine (Zantac) → more potent - less side effects →
6.6 mg / kg B.Wt 6-8 daily orally.
1.5 mg / kg B.Wt 6-8 daily IV.
- ☐ Should be continued for 10-20 days after clinical signs resolve.

2. Omeprazole (proton pump inhibitor)

- ☐ Block H/K ATPase pump → ↓ H ions secretion into lumen & HCL secretion from parietal cells.
- ☐ Long activity > 24 hrs.
- ☐ 4 mg/Kg B.wt orally every 24 hrs. for 14 days
- ☐ 0.5 mg/Kg B.wt once daily IV (↓ 90% of acidity).

2) Gastric antacid: given orally to neutralize stomach acid

1. Al hydroxide
2. Mg oxide & hydroxide 0.5 ml / Kg per os 6 times per day → ↑ PH > 3 but for 15-30 min SO must be given every 4 hrs.
3. Combination → 30 gm of Al hydroxide & 15 gm of Mg oxide & hydroxide → ↑ gastric pH.
4. Na bicarbonate.
5. Ca carbonate.

Side effects	
Al hydroxide	Ph depletion Ms weakness Bone resorption Hypercalciuria
Na bicarbonate	Na overload Systemic alkalosis
Mg oxide & hydroxide	Sever diarrhea Risk of renal failure
Ca carbonate	Hypercalcemia Renal impairment ++ of gastric secretion.

3) Mucosal Protectants: Sucralfate cheap – available – combined with ranitidine → 20 mg/kg B.wt per os every 8 hrs → mechanism

1. Adhere to ulcer & form pertinacious bandage.
2. ++ PGE2 & mucous secretion.
3. Inactivate pepsin & adsorb bile acids.

4) PG analogue (cyclo-oxygenase inhibitors) 1µg/kg BWt once daily ++mucous/bicarbonate barrier but produce S/E → abd.pain – bloat – cramps.

5) Intra gastric lavage with iced water & epinephrine (B-adrenergic VC) 8mg/500ml water

6) Fluid therapy